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Dietary factors are associated with coronary heart disease risk factors in college students

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ABSTRACT

It is hypothesized that healthy dietary and physical activity choices will be inversely associated with coronary heart disease (CHD) risk factors. Results from a cross-sectional study of 294 first-year University of Rhode Island students were used for the analyses. The presence of CHD risk factors was defined by the National Cholesterol Education Program Adult Treatment Panel III guidelines. Diet was assessed by three 24-hour food recalls, and physical activity was assessed by the International Physical Activity Questionnaire. Logistic regression models adjusted for sex estimated the odds of having CHD risk factors. A higher percent of kilocalories from alcohol was associated with a 9.9% increased risk for elevated triacylglycerol (odds ratio [OR], 1.099; 95% confidence interval [CI], 1.000–1.207). Sugar intake (OR, 1.015; 95% CI, 1.004–1.026), saccharin intake (OR, 1.047; 95% CI, 1.015–1.080), and body mass index (BMI; OR, 1.139; 95% CI, 1.037–1.252) were associated with an increased risk of low high-density lipoprotein cholesterol; dietary fiber intake (OR, 0.934; 95% CI, 0.873–1.000) was associated with a decreased risk of low high-density lipoprotein cholesterol. Participants with a higher BMI were 9.4% more likely to have elevated fasting glucose (OR, 1.094; 95% CI, 1.004–1.192) and 193.6% more likely to have a larger waist circumference (OR, 2.936; 95% CI, 1.543–5.586). Dietary factors and BMI are better indicators of CHD risk than physical activity is in this population.

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1. Introduction

Young adulthood is a critical transitional period, especially the first year of college, with most students living away from home for the first time, thus having increased independence [1]. Although college students, traditionally 18 to 24 years of age, are typically perceived to be healthy [2,3], the presence of traditional coronary heart disease (CHD) risk factors such as overweight (33%), low-density lipoprotein cholesterol (LDL-C;

53%), hypertension (47%) [4], elevated triacylglycerol (TAG) (18%), and low high-density lipoprotein cholesterol (HDL-C; 20%) [5] suggests otherwise. Lifestyle choices made during college, including lower diet quality and decreased physical activity compared with what high schoolers report [6], greatly influence these traditional CHD risk factors [7], which track forward to adulthood [8]. College students' diets exceed recommendations of total fat (46% vs 35% of total energy) and saturated fat (13% vs 10% of total energy) and lack whole

Abbreviations: 24HR, 24-hour recall; BMI, body mass index; BP, blood pressure; CHD, coronary heart disease; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; IPAQ-S, International Physical Activity Questionnaire Short Form; kcal, kilocalories; LDL-C, low-density lipoprotein cholesterol; MET-min/wk, metabolic equivalent-minutes/week; SBP, systolic blood pressure; TAG, triacylglycerol; TC, total cholesterol; WC, waist circumference.

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grains and fiber; only 43% of women and 51% of men meet the recommended intake [8]. More than 60% of college students do not engage in moderate or vigorous physical activity [9]. Poor diet and inactivity in college contribute to weight gain, which occurs 6 times faster for college students compared with young adults not in college [10]. This additional weight, most of which is excess body fat, leads to an increased CHD risk [11] via dyslipidemia and hypertension [7].

Previous studies in those 19 to 67 years of age have indicated that increased physical activity and diets that are in alignment with the *Dietary Guidelines for Americans* are predictive of a more favorable lipid profile, but few studies have examined the same relationships in young adults or college students [12–14]. Research including adolescents and young adults (12–35 years) reports that specific dietary components and body composition measures including sugar-sweetened beverages [15], saturated fat, monounsaturated fat, alcohol intake, waist-to-hip ratio [16], and body fatness [11] are significant predictors of CHD risk. However, these studies mainly looked at how individual diet or physical activity factors influence CHD risk in a wide range of ages rather than the combined influence of these factors in college students. To our knowledge, the only study that examined the predictive value of multiple lifestyle choices of college students (18–26 years) reported that smoking, binge drinking, lack of cardiovascular exercise, and a diet high in saturated fat were predictive of abnormal HDL-C concentrations and total cholesterol (TC)/HDL ratio [17]. Although Spencer [7] examined various lifestyle factors and CHD risk, the focus was fat and fiber without evaluating other dietary components and risk factors beyond blood pressure (BP), TC, HDL-C, and the TC/HDL-C ratio were not measured. Other factors including LDL-C, TAG, glucose, and waist circumference (WC) would also be important to examine because they are established, modifiable CHD risk factors.

Young adults underestimate the importance of a healthy diet and regular physical activity at this point in their life [18], and this may play a part in the poor lifestyle choices made in college. Therefore, the identification of dietary and physical activity factors that have the largest impact on CHD risk would provide a framework for the development of environmental and policy changes on campus. These changes could help motivate college students to make better choices to prevent disease progression. This cross-sectional analysis will be the first to thoroughly examine dietary intake with three 24-hour dietary recalls (24HR) and to measure multiple CHD risk factors including TC, LDL-C, HDL-C, TAG, glucose, BP, and WC in this population. The objective of this study is to use logistic regression models to determine how well lifestyle choices such as dietary intake and physical activity explain CHD risk factors present in this sample of college students. It is hypothesized that healthier dietary and physical activity choices will be inversely associated with CHD risk factors in this population.

2. Methods and materials

2.1. Participants

This was a cross-sectional study done at the University of Rhode Island with first-year college students. A convenience

sample of 294 first-year students were recruited from spring 2008 through fall 2009. Students between 18 and 24 years of age were eligible for the study. Exclusion criteria included being pregnant or lactating, or self-report of one of the following conditions: eating disorder, liver disease, bleeding disorder, diabetes, cancer, or CHD. Participants were recruited via classroom announcements, advertisements in the school newspaper, flyers, and word of mouth. All participants read and signed an informed consent form approved by University of Rhode Island's institutional review board.

2.2. Anthropometrics

Trained study staff performed all measurements using standard procedures. Each measurement was conducted in duplicate, unless variance between measurements exceeded the standard, in which case the measurement was repeated. The average of 2 readings was recorded. Height and weight were measured after participants voided, in light clothing and without shoes. Height was measured to the nearest 0.1 cm using a Seca 220 stadiometer (Seca Corporation, Hamburg, Germany). Weight was measured to the nearest 0.1 kg using a calibrated digital Seca 769 scale (Seca Corporation). Body mass index (BMI) was calculated using the following formula: weight in kilograms/height in meters². The following BMI categories were used: underweight (<18.5 kg/m²), normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), and obese (≥30.0 kg/m²) [19]. Participants' WC was measured at the top of the iliac crest upon exhalation to the nearest 0.1 cm using a Gulick fiberglass, nonstretchable tape measure with an attached tensometer (Patterson Medical, Mount Joy, PA, USA).

2.3. Biochemical measurements

For each subject, a trained phlebotomist obtained two 12-hour fasting venous blood draws on 2 nonconsecutive morning visits in the same week, which were 2 days apart. Briefly, plasma was obtained via centrifugation (Eppendorf Centrifuge 5810, Hamburg, Germany) of whole blood for 20 minutes at 2200 RPMs at 4°C. The following preservation cocktail was added to the plasma: 0.01 g/100 g of phenylmethylsulfonyl fluoride (Roche, Indianapolis, IN, USA), 0.01 g/100 g of sodium azide (Fisher, Fairlawn, NJ, USA), and 0.05 g/100 g of aprotinin (Fisher) [20]. Samples were stored in a –80°C freezer until analysis. Total cholesterol concentrations were determined via Roche Diagnostics Chol kit (Roche) [21]. A Roche/Hitachi Chol kit (Roche) was used for HDL-C analysis after dextran sulfate and magnesium chloride (Acros Organics, Morris Plains, NJ, USA) were used to precipitate out the apolipoprotein B containing lipoproteins [22]. Triacylglycerol concentrations were determined using a Roche/Hitachi Trig/GB kit (Roche) [23], and LDL-C concentrations were calculated using the Friedewald equation [24]. Plasma glucose concentrations were obtained using an Autokit Glucose (Wako Diagnostics, Richmond, VA, USA). All plates were read in a Biotek ELX 808 plate reader (Biotek, Winooski, VT, USA).

2.4. Blood pressure

Blood pressure was measured using previously described methods [25]. Briefly, after a 5-minute seated resting period, a

trained exercise physiologist measured each subject's resting BP on the right arm using a Littman Select stethoscope (Fisher Scientific) and a Welch-Allyn (Fisher Scientific) BP cuff appropriately sized for arm circumference. Blood pressure was conducted in duplicate, with a 1-minute interval between measurements. If variance between 2 measurements exceeded 2 mm Hg, the measurement was repeated. The average of 2 readings within the standard was used for analysis.

2.5. Dietary and physical activity measures

Twenty-four-hour dietary recalls were collected and analyzed by trained staff using the multiple-pass method in conjunction with the Nutrition Data System for Research software (University of Minnesota, Minneapolis, MN, USA) versions 2007-2009. This program uses a protocol that increases the accuracy of food descriptions, preparation, and additions [26]. All participants completed three 24HR—1 in-person and 2 over the telephone [27]—on 3 randomly selected, nonconsecutive days, including 2 weekdays and 1 weekend day, because this method has been shown to provide a good estimate of a person's usual intake [28]. Nasco food models (eNasco, Fort Atkinson, WI, USA) and food amount booklets were available during the initial in-person 24HR to facilitate visualizing portion sizes and obtaining accurate estimations of foods consumed [29]. Participants were given the booklets for the telephone 24HR. The mean values of the 3 recalls provided dietary data for analysis.

The International Physical Activity Questionnaire Short form (IPAQ-S) was used to estimate physical activity level [30]. The IPAQ-S is a valid and reliable tool to estimate physical activity in young adults [31]. Participants reported the frequency (days per week) and duration (time in minutes) of varying levels of physical activity over the previous 7 days. The IPAQ-S categorizes mode of activity by sitting, walking, moderate physical activity, and vigorous physical activity. Physical activity is expressed in metabolic equivalent minutes per week (MET-min/wk). The total amount of physical activity performed in a week was determined by calculating the sum of moderate, vigorous, and walking MET-min/wk. Metabolic equivalents were calculated using the scoring protocol, where 1 MET = resting energy expenditure, 3.3 METs = walking, 4 METs = moderate-intensity physical activity, and 8 METs = vigorous-intensity physical activity [32].

2.6. Statistical analyses

Continuous variables are displayed as means and SDs and categorical data as numbers and percentages. Differences between sexes on continuous and categorical variables were analyzed by independent-samples *t* tests and χ^2 tests, respectively. For logistic regression analyses, the CHD risk factors were dichotomized into presence or absence of risk, with presence defined as follows: TC greater than 200 mg/dL, LDL-C greater than 100 mg/dL, HDL-C less than 40 for men and less than 50 for women, TAG greater than 150 mg/dL, glucose greater than 100 mg/dL, systolic BP (SBP) greater than 130 mm Hg, diastolic BP (DBP) greater than 85 mm Hg, WC greater than 88 cm for women and greater than 102 cm for men [7]. Univariate logistic regressions were completed to show the

strength of the dietary and physical activity variables as predictors of the individual CHD risk factors. Sex was controlled for all risk factors, except for TC and DBP, because no men had high TC and no women had high DBP. Systolic BP was not examined because no participants met this criterion. Predictors significant at $P < .10$ were then entered into multivariate logistic regressions, again controlling for sex when possible. SPSS software (SPSS, Inc, Chicago, IL, USA) version 19.0 was used for analysis. Significance was set a priori at $P < .05$ for multivariate logistic regression, *t* tests, and χ^2 tests.

3. Results

Tables 1 and 2 display participant characteristics. Thirty-three participants withdrew (11.2% attrition) because of a lack of time or interest ($n = 16$), illness ($n = 6$), discomfort with blood draws ($n = 4$), involvement in a dietary intervention study ($n = 2$), or unknown reasons because they were nonresponsive to all communications from study staff ($n = 5$). A final sample of 261 participants, 85 men (32.6%) and 176 women (67.4%), completed all required study assessments. As expected, men had significantly greater height, weight, WC, and kilocalorie (kcal) intake than women ($P < .05$). Eighty-two percent of the participants self-reported being white; 4.6%, African American; 4.6%, Asian; 0.4%, American Indian/Alaska Native; and 8.4%, other. Of this group, 10.7% identified themselves as Hispanic/Latino. The mean (SD) age was 19.3 (1.4) years. Six percent of participants were nutrition and dietetics majors, 26% were allied health majors (pharmacy and nursing), 8% were kinesiology majors, and 60% were other majors.

As previously stated, independent variables associated with CHD risk factors at the $P < .10$ level in univariate logistic regressions were entered into the multivariate model. Using these parameters, no significant results were found for TC, SBP, or DBP. Percent of kcal from alcohol (odds ratio [OR], 1.112; 95% confidence interval [CI], 1.019-1.213), percent of kcal from polyunsaturated fatty acids (OR, 0.826; 95% CI, 0.675-1.012), and folate (OR, 1.001; 95% CI, 1.000-1.002) were associated with elevated TAG. Alcohol remained significant in the multivariate model; those with a higher percentage of calories from alcohol were 9.9% more likely to have elevated TAG (OR, 1.099; 95% CI, 1.000-1.207).

Percent of kcal from carbohydrate (OR, 1.055; 95% CI, 1.006-1.107), fat (OR, 0.949; 95% CI, 0.895-1.007), and monounsaturated fatty acids (OR, 0.882; 95% CI, 0.766-1.014) were associated with decreased HDL-C concentrations for sex. Dietary cholesterol (OR, 0.996; 95% CI, 0.994-0.999), dietary fiber (OR, 0.951; 95% CI, 0.901-1.006), sugars (OR, 1.006; 95% CI, 0.999-1.014), saccharine (OR, 1.026; 95% CI, 1.002-1.051), moderate physical activity (OR, 0.999; 95% CI, 0.998-1.000), vigorous physical activity (OR, 1.000; 95% CI, 0.999-1.000), and BMI (OR, 1.119; 95% CI, 1.035-1.210) were also associated with low HDL-C for sex. In the multivariate model, higher sugar intake (OR, 1.015; 95% CI, 1.004-1.026), saccharin intake (OR, 1.047; 95% CI, 1.015-1.080), and BMI (OR, 1.139; 95% CI, 1.037-1.252) were associated with a 1.5%, 4.7%, and 13.9% increased risk of low HDL-C, respectively; dietary fiber intake (OR, 0.934; 95% CI, 0.873-1.000) was associated with a decreased risk of

Table 1 – Participant characteristics

Characteristics	All (n = 261)	Male (n = 85)	Female (n = 176)	P
Age (y)	18.3 ± 0.5	18.3 ± 0.6	18.3 ± 0.5	.368
Height (cm)	168.1 ± 8.8	176.6 ± 6.8	164.0 ± 6.3	<.001*
Weight (kg)	66.0 ± 13.2	75.6 ± 13.1	61.4 ± 10.5	<.001*
BMI (kg/m ²)	23.3 ± 3.8	24.2 ± 3.7	22.9 ± 3.8	.009*
WC (cm)	78.6 ± 9.5	81.9 ± 8.6	77.0 ± 9.5	<.001*
TC (mg/dL)	148.7 ± 27.4	137.8 ± 21.2	154.0 ± 28.5	<.001*
LDL-C (mg/dL)	73.0 ± 25.5	69.8 ± 23.0	75.0 ± 26.0	.118
HDL-C (mg/dL)	57.3 ± 13.6	52.1 ± 11.7	59.8 ± 13.9	<.001*
TAG (mg/dL)	93.4 ± 49.8	79.4 ± 42.2	100.1 ± 51.9	.001*
Glucose (mg/dL)	89.2 ± 8.7	89.5 ± 8.4	89.1 ± 8.9	.688
SBP (mm Hg)	105.0 ± 10.6	109.3 ± 10.9	102.9 ± 9.9	<.001*
DBP (mm Hg)	66.8 ± 7.3	69.2 ± 8.4	65.6 ± 6.5	.001*
Energy intake (kcal)	2095.2 ± 726.4	2698.8 ± 824.2	1803.7 ± 443.0	<.001*
% kcal from carbohydrate	52.7 ± 7.3	49.6 ± 6.6	54.3 ± 7.1	<.001*
% kcal from protein	15.2 ± 3.6	16.7 ± 3.6	14.5 ± 3.4	<.001*
% kcal from fat	31.3 ± 5.9	33.2 ± 6.1	30.4 ± 5.6	<.001*
Physical activity (total MET-min/wk)	3778.2 ± 3188.0	4462.0 ± 3373.6	3448.0 ± 3049.6	.001*

Data expressed as means ± SDs.

* Sex differences determined by independent-samples t tests, $P < .05$.

low HDL-C for sex. Vigorous physical activity (OR, 1.000; 95% CI, 0.999-1.000) approached significance for predicting HDL-C. Multivariate logistic regression was not done on LDL-C because iron intake was the only variable significantly associated with LDL-C in univariate models ($P < .01$).

Percent of kcal from saturated fatty acids (OR, 1.139; 95% CI, 0.977-1.327), folate (OR, 0.998; 95% CI, 0.996-1.000), vigorous physical activity (OR, 1.000; 95% CI, 0.999-1.000), and BMI (OR, 1.097; 95% CI, 1.008-1.195) were associated with elevated fasting glucose. Only BMI remained significant in the multivariate model (OR, 1.094; 95% CI, 1.004-1.192); those with a higher BMI were 9.4% more likely to have elevated fasting glucose.

Total kcal (OR, 0.999; 95% CI, 0.998-1.000), percent of kcal from carbohydrate (OR, 1.149; 95% CI, 1.007-1.312), sugars (OR, 0.987; 95% CI, 0.974-1.000), added sugars (OR, 0.980; 95% CI, 0.963-0.997), aspartame (OR, 1.006; 95% CI, 0.999-1.012), sucralose (OR, 1.001; 95% CI, 1.000-1.001), moderate physical activity (OR, 0.998; 95% CI, 0.996-1.000), total physical activity (OR, 1.000; 95% CI, 1.000-1.000), and BMI (OR, 2.239; 95% CI, 1.594-3.143) were associated with a larger WC for sex. As expected, those with a higher BMI were 193.6% more likely to

have an elevated WC for sex in the multivariate model (OR, 2.936; 95% CI, 1.543-5.586).

5. Discussion

This study is the first to examine the combined influence of dietary and physical activity factors on traditional CHD risk factors in college students. The results from this study support our hypothesis and indicate that healthy dietary patterns among college students were significantly associated with lower TAG, glucose, and WC and higher HDL-C. Healthy dietary patterns such as increased fiber intake, a lower BMI, and decreased consumption of sugar, saccharine, and alcohol were associated with significantly less CHD risk in this population. Unexpectedly, physical activity was not significantly associated with any of the CHD risk factors in multivariate models.

The findings extend previous results that indicated that HDL and TC/HDL ratio were best predicted by smoking, exercise, binge drinking, and saturated fat intake [17]. Other studies done in older populations confirm that healthy dietary and physical activity choices are associated with a decreased risk of CHD [33], stroke [34], and lower concentrations of LDL-C, TC, and TAG [14].

Body mass index was most strongly associated with CHD risk factors, and dietary components were better predictors in this population than physical activity patterns. Physical activity was significantly associated with glucose, HDL-C, and WC in univariate models. However, ORs of 1.00 indicated that there was no reduction in risk and associations became nonsignificant in multivariate models. Although the ability of physical activity to reduce the risk of CHD is well established [35-37], a similar study that examined multiple lifestyle factors in an adult population found that physical activity was not significantly associated with cardiometabolic risk [13]. The absence of an association may be attributed to the reliance on self-reported physical activity because of the

Table 2 – Prevalence of CHD risk factors

Risk factor	Male (n = 85), n (%)	Female (n = 176), n (%)	P
WC (>88 cm for women; >102 cm for men)	3 (3.6)	14 (8.0)	.283
TC (>200 mg/dL)	0 (0)	14 (8.0)	.006*
LDL-C (>100 mg/dL)	7 (8.2)	25 (14.2)	.227
HDL-C (<40 mg/dL for men; <50 mg/dL for women)	7 (8.2)	39 (22.2)	.005*
TAG (>150 mg/dL)	7 (8.2)	30 (17.0)	.060
Glucose (>100 mg/dL)	9 (10.6)	21 (11.9)	.838
DBP (>85 mm Hg)	4 (4.7)	0 (0)	.011*
SBP (>130 mm Hg)	0 (0)	0 (0)	–

* Sex differences determined by χ^2 tests, $P < .05$.

lack of a standardized method for the assessment of physical activity [38]. Self-administered questionnaires that require participants to estimate physical activity over the last 7 days are subject to overestimation of time spent in high-intensity activities and underestimation of time spent in light- and moderate-intensity activities [39,40]. In the future, physical activity measures should include more objective methods of assessing physical activity such as pedometers or accelerometers.

Regression models were not constructed for SBP, DBP, and TC because there was only 1 subject with an elevated SBP, no women had elevated DBP, and no men had elevated TC. A recent study by Burke et al [4] reported that 86% of college students aged 18 to 24 years had either elevated SBP or elevated DBP and 27% had elevated TC. The difference in prevalence rates could be explained by the use of different cut points, as the present study defined the presence of risk as BP of at least 130/85 mm Hg (National Cholesterol Education Program Adult Treatment Panel III), whereas Burke et al used BP of at least 120/80 mm Hg.

The lack of association between dietary components and LDL-C was unexpected. Dietary patterns low in saturated fat, trans-fat, and cholesterol are associated with higher concentrations of LDL-C [41]. The only significant association that existed in a univariate model for LDL-C was iron; therefore, multivariate analysis was not performed for LDL-C.

A higher percentage of kcal from alcohol resulted in a 9.9% increase in risk for elevated TAG. Because most of the participants were underage and may have feared reporting alcohol consumption, the 9.9% increase in risk may be a conservative estimate.

A strength of this study was the use of the multiple-pass method in conjunction with Nutrition Data System for Research. Another strength is the use of a 3-day average for all diet analyses. In addition, all anthropometric measurements were collected by trained staff using standard protocol and did not rely on self-report. Several limitations, however, should be considered. Owing to the cross-sectional nature of this study, causality cannot be inferred. Also, the study population was predominantly white and female, limiting the extent to which results can be generalized to other populations.

The presence of CHD risk factors among college students, along with the development of lifestyle choices at college, makes this population a prime target for CHD prevention efforts. College campuses are an ideal setting to reach 42% of the young adult population [42] and to convey the importance of CHD risk reduction through diet and exercise. Furthermore, although college students indicate that they are aware of CHD risk, their behaviors often do not reflect their knowledge [43]. Environmental and policy changes are needed on campus to facilitate behavior change and guide students toward making better dietary and physical activity choices.

Relatively small changes in the physical environment can produce behavioral changes [44]. For example, placing healthy foods in more prominent places, removing trays from dining halls, and using point-of-selection signage [45] are inexpensive ways to prompt healthier dietary choices. Other changes such as using point-of-choice prompts for stair climbing [46], decreasing prices for exercise classes, and increasing the attractiveness of recreational facilities

can be implemented to increase physical activity [44]. Students, health professionals, policy makers, dining hall managers, and restaurant owners should work together to create an environment that supports the adoption of lifelong healthy behaviors.

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